THE FORENSIC PANEL
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Re: Robert T. Brockman

August 6, 2021

Dear Mr. Loonam,

At your request, I reviewed the available diagnostic imaging studies of Robert Brockman, an 80-year-old defendant who has been worked up for cognitive problems since 2018. Mr. Brockman is charged in a complex indictment with details and history that require him to be actively engaged in informing his attorneys with reliable and valid information, to be making decisions, and to be guiding the attorneys through records and evidence for which they can only partly inform their preparation. His capacity to inform his attorneys and to engage the mental and physical rigors of trial is in question, and a court hearing is anticipated.

Mr. Brockman's diagnosis is partly in dispute. An extensive workup from different highly reputable neuroscience specialists at Baylor University School of Medicine has established a diagnosis of Parkinson's disease. Mr. Brockman has also been diagnosed with dementia, based on history, functional decline, and his performance on neuropsychological testing.

Recently, court-appointed specialists in psychiatry, neuropsychology, and neurology have submitted reports to the court, after conducting their own examination. The three examiners opined that Mr. Brockman 1) at worst has Mild Cognitive Impairment 2) is malingering his neurocognitive disease 3) that the course of his condition was expected to fluctuate, with the potential for improvement and even normalization during some periods.

A number of neuroimaging studies have informed the case in recent months. The matter was therefore referred for my review through The Forensic Panel in order to ascertain:

What does the recent neuroimaging in this case inform about the nature and severity of Mr. Brockman's diagnosis?

SOURCES OF INFORMATION

1) Robert Brockman Indictment, October 1, 2020
2) Declaration of Dr. James Pool, MD., November 25, 2020

3) Declaration and exhibits of Kathryn Keneally, December 8, 2020
4) Declaration of Peter J. Romatowski, December 8, 2020
5) Draft declaration of Ryan Darby, February 28, 2021
6) Dr. Jankovic's report of office visit, January 30, 2019
7) MRI results and images, November 2, 2018
8) Diagnostic Report re: NM Datscan, Brain SPECT, February 14, 2019
9) Dr. Michele York Neuro-psychological evaluation, March 1, 2019
10) Dr. Yu Notes from Mr. Brockman's appointment, March 20, 2019
11) Report of Dr. Pool's Annual Physical, October 1, 2019
12) Dr. York Forensic Evaluation, December 3, 2019
13) Dr. Pool's examination, October 5, 2020
14) Dr. York's neuropsychological exam, October 7, 2020
15) Dr. Denney test data, May 19, 2021
16) Dr. York test data, March 1, 2019, December 3, 2019, October 7, 2020
17) Methodist hospital records for infectious disease, May 31-June 11, 2021
18) Methodist hospital records for infectious disease, March 15-19, 2021
19) Email between Dr. Yudofsky and Mr. Brockman regarding memory problems, May 3-4, 2017
20) Dr. Yudofsky consultation notes, October 20, 2018 – October 23, 2020
21) PET scan results, March 12, 2021
22) Peer oversight call with Thomas Guilmette, Ph.D., Michael Welner, M.D., Christopher Whitlow, M.D., Marc Agronin, M.D., Timothy Shepherd, Ph.D., M.D., July 30, 2021
23) Amyloid PET scan results and images, July 28, 2021
24) Brain MRI and volumetric results and images, July 30, 2021

NEUROIMAGING DATA CONSIDERED AND COMPARED

1. Brain MRI scan dated November 2, 2018

The interpreting radiologist reported: "No intracranial abnormalities, particularly no disproportionate lobar atrophy." I have reviewed this imaging study and agree with this interpretation, though do appreciate diffuse cerebral volume loss.

2. Single-photon emission computed tomography (SPECT) scan with ^{123}I-ioflupane for dopamine transporter imaging (DaTscan) dated February 14, 2019

The interpreting radiologist reported: "Severe loss of dopaminergic neuronal function in the bilateral dorsal striata with loss greater on the right compared to the left." I have reviewed this DatScan study and agree with this interpretation.

3. Positron emission tomography-computed tomography (PET-CT) scan with ^{18}F-fluorodeoxyglucose (FDG) dated March 12, 2021
The interpreting radiologist reported: "Mildly reduced uptake in the right parietal lobe. Findings are very mild, but suggestive of early neurodegenerative disease, either Alzheimer's disease or dementia with Lewy bodies (Parkinson's disease with dementia). Findings are unlikely to represent frontotemporal dementia." I have reviewed this study and agree with this interpretation.

4. Positron emission tomography (PET) scan with ^{18}F-florbetapir (Amyvid) dated July 28, 2021
The interpreting radiologist reported: "Positive study, indicating moderate to frequent amyloid neuritic plaques." I have reviewed this study and agree with this interpretation.

5. Brain magnetic resonance imaging scan dated July 30, 2021
The interpreting radiologist reported: "Moderate diffuse cerebral volume loss with proportional ventricular prominence. Mild chronic microvascular ischemic change." I have reviewed this study and agree with this interpretation. Upon comparison with brain MRI dated November 2, 2018, I also note that the cerebral volume loss and changes of chronic microvascular ischemic disease have progressed.

6. Neuroreader report derived from brain MRI scan July 30, 2021
The following brain matter volumes with a percentile lower than 25% and all cerebrospinal fluid structures with a percentile over 75%: Right ventral diencephalon (24.80%), Temporal lobe (23.80%), Right temporal lobe (21.94%), right lateral ventricle (79.15%).

FORENSIC NEURORADIOLOGY ASSESSMENT

The 2019 DaTscan demonstrates decreases in bilateral dorsal striata dopamine transporter, which is diagnostic of Parkinson's disease. Mr. Brockman has Parkinson's disease.

Mr. Brockman's brain MRI demonstrates cerebral volume loss that has progressed since 2018, with diencephalon and temporal lobe volume that is lower than the 25th percentile according to an FDA approved volumetric analysis. Brain volume this low would be more compatible with neurodegenerative diseases associated with cognitive dysfunction and dementia rather than mild cognitive impairment.

Mr. Brockman's FDG PET scan revealed decreases in metabolism in parietal lobe, also suggestive of a neurodegenerative process, with Alzheimer's disease or dementia with Lewy bodies (Parkinson's disease with dementia) as primary considerations for this finding. His Amyvid PET scan was positive for amyloid, which is an abnormal protein that accumulates in patients with Alzheimer's disease and with Lewy body dementia, as well as other neurodegenerative diseases. Taken together:

Mr. Brockman has Parkinson's disease, with:

1) brain volume loss that is profound in the temporal lobes that mediate important cognitive functions like learning and memory.

2) decreased brain function as reflected by low metabolism in the parietal lobe.

3) accumulation of abnormal brain amyloid.

The confluence of the multiple complementary imaging findings across multiple imaging domains taken together represent objective data supportive of a diagnosis of dementia. Taken together with the degree of dysfunction consistently demonstrated by neuropsychological testing, along with Mr. Brockman's functional decline, these imaging findings would be expected.

In this regard, the evidence of dementia from Mr. Brockman's neuroimaging identifies the structural and functional abnormalities that explain the significant findings noted in clinical interviewing and psychological testing. Volume loss/atrophy in temporal lobe specifically would be expected in a patient with Mr. Brockman's poor performance on cognitive testing.

The temporal pathology is a common objective imaging finding in patients with memory loss and reflects an anatomical pattern of neurodegenerative disease that affects memory and cognitive function, such as Alzheimer's disease and Lewy body dementia.

The progressive volume loss on MRI along with the amyloid positivity and the magnitude of the volumetric loss in the temporal lobe is beyond what would be expected for Mild Cognitive Impairment and makes the diagnosis of dementia all the more likely.

The degree of volume loss combined with the functional brain abnormalities and amyloid burden would be expected to impair Mr. Brockman's physiologic resilience and at high risk for delirium secondary to infection, which is a common scenario for patients with underlying neurodegenerative conditions.

The magnitude of visually obvious volume loss on MRI is beyond anything that would be reversible, with temporal lobe (an important structure in cognitive functions like memory) being well within the bottom 25% of the population for volume based on the quantitative analysis. The volumetric brain changes on MRI between 2018 and 2021 are reflective of acceleration of the brain degenerative process.

In the research domain, we frequently conduct MRI structural (volumetric), PET functional (FDG-PET) and PET molecular (Amyvid-PET) studies to characterize a data

driven "temporal-parietal meta-region of interest (ROI)" that is usually susceptible to age-related neurodegenerative disease, including typical Alzheimer's disease (Nelson et al. 2019).

Temporal-parietal brain has been consistently among the top performing regions of interest in brain region-specific studies discriminating between amyloid PET negative cognitively unimpaired and amyloid PET positive cognitively impaired individuals in the Mayo Clinic Study of Aging and Mayo Alzheimer Disease Research Center (Schwarz et al. 2016; Jack et al. 2017).

In summary, these imaging findings are suggestive of a progressive neurodegenerative process and support Mr. Brockman's cognitive testing results, raising concern for a neurodegenerative disease with dementia, which include common causes like Parkinson's disease, Parkinson's disease with Lewy body dementia and Alzheimer's disease. Given what we know about structural and functional abnormalities in temporal-parietal brain regions, there is compelling objective evidence that an underlying neurodegenerative process is ongoing for Mr. Brockman, with Alzheimer's dementia representing a very strong possibility.

Thank you for the opportunity to review this matter. Do reach out to me with any new imaging or other pertinent study for my review.

Sincerely yours,

Christopher T. Whitlow, MD, PhD, MHA

References:

Nelson PT, Dickson DW, Trojanowski JQ, et al. Limbic-predominant age-related TDP-43 encephalopathy (LATE): consensus working group report. *Brain*. 2019;142(6):1503-1527.

Schwarz CG, Gunter JL, Wiste HJ, et al. A large-scale comparison of cortical thickness and volume methods for measuring Alzheimer's disease severity. *Neuroimage Clin*. 2016;11:802-812.

Jack CR, Jr., Wiste HJ, Weigand SD, et al. Age-specific and sex-specific prevalence of cerebral β-amyloidosis, tauopathy, and neurodegeneration in cognitively unimpaired individuals aged 50-95 years: a cross-sectional study. *Lancet Neurol*. 2017;16(6):435-444.